Thyroid-Associated "mRNA X" Deficiency Results in Epigenetic Changes in Mitochondrial Metabolism Throughout Body Resulting in Oxygen-Lean Manufacturing Process for ATP; Accounts for Ability of Pituitary to Know When to Manufacture Additional TSH

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Introduction

Although ATP is chemically identical regardless of mode of production, glucose, with its six oxygens, may independently provide sufficient oxygen to produce ATP anaerobically if the mitochondria's process for handling glucose is altered. All six oxygens, some of which may have otherwise been discarded in the ATP production process, are all used in this mode of ATP production.

Abstract

Although this difference, if its existence is confirmed, would not bestow an aerobic lifeform with the ability to survive without oxygen, the epigenetic changes to the mitochrondria caused by this type of as-yet unidentified dimension of hypothyroidism mimic those changes that would be observed in a lifeform that has been forced to adapt to reduced oxygen levels.

The result is that an individual lacking in the as-yet unidentified "thyroid hormone X;" more than likely not a hormone but an mRNA; would have an altered mitochondrial metabolism that more efficiently utilizes oxygens in glucose molecules, eschewing available oxygen from hemoglobin. The net result would be a patient that has reduced body temperature (as less O2 is being metabolized relative to glucose) and that a patient's weight is hyperstabilized regardless of T4 supplementation.

When T4 is supplemented, the thyroid is freed to produce the "X" mRNA in greater quantities as it may focus its efforts entirely on non-T4/T3 production. I furthermore would speculate that the mRNA responsible for this is the same one that a deficit of which results in the pituitary overproducing TSH. While the current assumption in this field is that it must be the presence of an mRNA that causes this TSH production (or perhaps the absence of T4) I would suggest that it is, in fact, the absence of an mRNA that is to blame. Furthermore, the very same mRNA a deficit of which spurs TSH production results in these epigenetic changes to mitochondria, resulting in a more efficient mode of oxygen reclamation from glucose molecules, reducing overall O2 use.

The confirmation of this hypothesis would go a long way toward explaining the many reported cases of hypothyroidism that present with elevated TSH but normal T4 and T3 levels. In these patients, weight loss is not reported despite T4 supplementation. Symptoms of more severe thyroid deficiency such as dangerous hypothermia seem to be resolved by T4 supplementation, suggesting that perhaps it is not T4 or T3 that restores (at least partially) normal metabolism, but rather, this benefit is an indirect result of the thyroid

being able to dedicate itself to the production of an entirely different, heretofore unidentified mRNA. The hormone may not be a hormone in the classical sense but may instead be an mRNA signal that controls mitochondrial gene expression. This may account for the failure of the scientific community to identify this mechanism to date.

Confirmation of this hypothesis may lead to a therapy that enables weight loss in patients with hypothyroidism who have reported experiencing difficulty in losing weight.

Once mitochronial gene expression/function has been altered as a result of actual (or perceived) chronic hypoxia, there is no extant mechanism for throwing all of the epigenetic switches back to their original position.

You may be wondering why, if the thyroid is actually an oxygen use-regulating organ (a paradigm shift in and of itself,) why the mitochondria becoming more efficient at using oxygen from glucose would result in weight retention.

The answer has to do with classical pulmonary respiration and Oxygen-CO2 exchange. An individual who has a more efficient oxygen metabolism (perhaps resulting from historical thyroid dysfunction) would not need to breathe as deeply (or exhale as completely) as an individual without these epigenetic changes. Shallow breathing results in reduced expulsion of CO2 and thus, their chronically higher serum CO2 levels result in weight retention. In cases of persistent thyroid-associated weight gain, rigorous aerobic exercise i.e. it involves heavy breathing, resolves the weight retention issues reported by those individuals.

Conclusion

I can therefore conclude that by restoring the default mode of mitochondrial oxygen metabolism (by forcing the mitochondrial epigenetic profile back to its original configuration,) the patient would be forced to inhale and exhale more deeply even when not exercising (something which cannot be consciously controlled) and would as a consequence experience weight loss. While their ability to hold their breath while under water would no longer be enhanced, they would likely be content with this trade-off.